An Unusual Case of Hyperkalemia during Hyperbaric Oxygen Therapy for Carbon Monoxide Poisoning

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Abstract

Carbon monoxide (CO) is a potentially lethal gas if inhaled that may present with a wide spectrum of symptoms non-specific to the degree of exposure. CO has an affinity for hemoglobin that is 240 times that of oxygen, forming carboxyhemoglobin (COHB). COHB does not transport oxygen and may produce a hypoxic state resulting in end-organ damage and significant long term neurological and affective sequel. High flow oxygen therapy forms the cornerstone of immediate therapy which may be hastened with hyperbaric oxygen therapy (HBO). HBO is not without side effects. Various adverse effects have been described but hyperkalaemia occurring during HBO has not yet been reported. We describe an unusual case of hyperkalaemia occurring during HBO for CO poisoning.

Keywords: Hyperkalemia; Carbon monoxide poisoning; Carboxyhaemoglobin; Hyperbaric oxygen

Introduction

Carbon monoxide is a toxic, colourless and odourless gas that is produced during incomplete combustion of organic fossil fuels. Symptoms of acute poisoning vary widely from mild neurological symptoms including dizziness, headaches and flu-like symptoms [1] to more serious effects mainly affecting the central nervous and cardiac systems. We present a rare case of hyperkalaemia that resulted during hyperbaric oxygen treatment and discuss the implications of both carbon monoxide poisoning and various possible causes of hyperkalaemia in this case.

Hyperkalaemia is defined as a serum potassium concentration greater than 5.5 mEq/L in adults. Levels higher than 7 mEq/L can lead to significant cardiovascular and neurological consequences. The aggressiveness of therapy is directly related to the rapidity with which hyperkalaemia has developed, the absolute level of hyperkalaemia, and the evidence of toxicity. In severe cases, treatment focuses on immediate stabilization of the myocardial cell membrane, rapid shifting of potassium to the intracellular space, and total body potassium elimination. In addition, all sources of exogenous potassium should be immediately discontinued. Indicators for more aggressive oxygen therapy include the rapidity and extent of hyperkalaemia that develops [2].

Case Report

A 39 year old, unauthorized immigrant was brought to our accident and emergency department after being found unconscious by other tenants who were sleeping in adjacent rooms. The patient shared a room with another man who was pronounced dead on site. A smouldering charcoal stove was found in the windowless room though no further smell was detected by paramedics. The patient had no known medical history and did not use any illicit drugs. Friends of the patient and the deceased claimed that the charcoal stove had been lit the previous night to warm the room.

At accident and emergency the patient had a Glasgow coma scale (GCS) of 7 (M2, V2, E3), a heart rate of 120 bpm, a blood pressure of 110/60mmHg and a temperature of 35.6 degrees Celsius. Clinical examination was unremarkable except for coarse crepitations of the left lung upon auscultation. The patient's decreased level of consciousness together with the presence of a smouldering charcoal stove pointed to a likely case of CO poisoning. This was confirmed by arterial blood gas analysis which showed a COHB level of 37% (using Nova ® biomedical pHOx Ultra TM). Normal values in healthy non-smokers adults should read less than 3% and up to 10-15% of COHB in smokers found in blood.

Subsequently the patient was intubated, ventilated and kept sedated with a propofol infusion. Five grams of hydroxycobalamin were administered in case of concomitant cyanide poisoning (later confirmed to be absent). The patient was treated with 100% oxygen therapy followed by hyperbaric oxygen therapy at a pressure of 2500 mmHg for the next 2 hours and 45 minutes. The patient remained haemodynamically stable throughout.

Radiological investigations included a normal chest X-ray (Figure 1) but CT-thorax (Figure 2) performed in view of...
hypoxaemia showed a left lower lobe collapse with material in the left lower lobe bronchus and consolidations in the posterior segment of the right lower lobe. This was postulated to be due to an aspiration during the period of unconsciousness and the patient was started on intravenous antibiotics.

The patient was kept intubated, ventilated and sedated for a period of 48 hours. An MR brain 48 hours after initial admission showed no radiological evidence of brain injury and after a trial off sedation and gradual weaning of ventilatory support the patient was successfully extubated. Though lethargic, the patient responded appropriately with no evidence of immediate neurological sequel. Response to intravenous antibiotics was also appropriate and the patient was transferred to a general ward for further recovery after 5 days. The Patient continued to improve and was discharged from hospital after nine days.

Discussion

Mechanisms of CO toxic effects are complex and lead to hypoxia of body tissues. CO binds avidly and reversibly to metallo-proteins found in hemoglobin with an affinity approximately 240 times greater than that of oxygen. This reduces the oxygen carrying capacity of hemoglobin and shifts the oxygen-hemoglobin curve to the left, impairing oxygen release at the tissue level [3]. In addition to binding to hemoglobin, 10–15% of CO binds to other proteins, particularly myoglobin within cardiac muscle, interfering with its function as an oxygen reservoir [4,5]. CO also exerts its toxic effects at a cellular level by binding to cytochrome oxidase in mitochondria, interfering with the electron transport chain and resulting in impairment of cellular respiration and formation of free radicals.

The presentation and physiological effects of CO poisoning are varied. Our case was brought to casualty in an unconscious state and following a high degree of clinical suspicion the diagnosis was confirmed by measurement of COHb levels within the blood. Symptoms may arise immediately or follow an asymptomatic period. Lucid intervals of up to 240 days have been observed after CO poisoning, although the mean latency for development of cognitive and behavioral symptoms is three weeks [6].

Multiple hypotheses explain the mechanism by which CO toxicity may lead to loss of consciousness and cerebral injury. There are acute and delayed neuropathological changes related to direct CO toxicity. Neurotoxicity may be secondary to a massive release of excitatory amino acids, particularly glutamate [7]. CO also activates neutrophils that produce reactive O₂⁻ species and cause brain lipid peroxidation. Peroxidation leads to the degradation of unsaturated fatty acids and the reversible demyelination of central nervous system lipids. CO increases the production and deposition of peroxynitrite (a potent oxidant) within blood vessel endothelium and brain parenchyma, leading to vascular compromise and cell death in neurons and neuronal cell lines [8]. Reoxygenation injury may also occur secondary to the production of partially reduced oxygen species, created during HBO. Oxygen species can oxidize essential proteins and nucleic acids, creating injury similar to reperfusion damage [7].

COHb concentrations do not always determine the severity of toxic damage at the level of selected organs, or serve as a prognostic index [9]. Our case presented with an initial COHb concentration of 37% but was found not to have suffered immediate neurological sequel after extubation and subsequent supportive therapy within the ward.

CO poisoning is also associated with cardiovascular effects which include myocardial ischemia, pulmonary edema, arrhythmia, and stunned myocardium syndrome [10]. Our case had successive raised troponin levels, compatible with myocardial ischaemia.

| Table 1: ABG values on admission to the intensive therapy unit at a FiO₂ of 60%. Hyperkalemia was treated according to local protocol consisting of calcium gluconate and insulin/dextrose infusion. |
|-----------------------|-----------------|
| ABG values            |                 |
| PaO₂                  | 64 mmHg         |
| PaCO₂                 | 46 mmHg         |
| pH                    | 7.303           |
| HCO₃⁻                 | 23 mmHg         |
| Lactate               | 1.7             |
| BE                    | -3.2            |
| Na⁺                   | 138 mmHg        |
| Cl⁻                   | 111 mmHg        |
| K⁺                    | 7.8 mmHg        |

This case shows an unusual increase in serum potassium levels (serum potassium of 7.9 mmol/L) which occurred during treatment with HBO. Initial hyperkalemia (potassium ≥ 5.1 mmol/L) has been found to be associated with an increased likelihood of an unconscious state on arrival;[11] however, on arrival our patient presented with a serum potassium level of 3.73 mmol/L. Hyperkalemia was treated according to local protocol consisting of calcium gluconate and insulin/dextrose infusion (Table 1). The cause for this elevated serum potassium rise was not due to rhabdomyolysis and acute renal failure, a rare but known complication of CO poisoning. This diagnosis was ruled out by the low serum creatinine kinase levels. It is also unlikely to be due to administration of suxamethonium during intubation as normal muscle releases enough potassium during succinylcholine-induced depolarization to raise serum potassium by around 0.5 mEq/L in non-renal failure patients [14]. The patient was on no known regular medications. Platelet count on admission was of 305 X 10⁹/L (146 – 302 X 10⁹/L) thus making pseudohyperkalemia due to thrombocytosis an unlikely cause. Hemoglobin value on admission was 15.7 g/Dl (14.1-17.2 D/Dl) and remained stable throughout thus excluding hemolysis. Initial high sensitivity troponin T levels were raised at 325 ng/L and 656 ng/L (3-14 ng/L) however no changes were noted on EKG (Table 2).

An abnormal transthoracic echocardiogram excluded any regional wall abnormality. Hyperkalemia secondary to an acute myocardial event was deemed to be unlikely with raised troponin T levels attributed to catecholamine release and increased myocardial load secondary to decreased oxygen supply to the heart. We postulate that in this case hyperkalemia during HBO occurred due to reperfusion injury caused by the production of partially reduced oxygen species created during HBO.

The treatment of CO poisoning rests on the rapid restoration of oxygenation to bodily organs. The elimination half-life of CO is 4–5 hours; however, with the administration of 100% O₂ via a tight-fitting face mask at normal atmospheric pressure the half-life can be reduced to 1 hour [12]. HBO involves the administration of 100% oxygen in a pressurized chamber. It further hastens the reversal of the binding of CO binding to hemoglobin and to myoglobin and can provide oxygen to tissues independent of hemoglobin [13]. When administered at 2.5 atm absolute pressure, HBO decreases the elimination half-life of CO to 20 minutes. In addition to restoring tissue oxygenation, HBO appears to improve mitochondrial function, to alter inflammatory responses induced by CO, and to reduce post ischemic brain damage in those exposed to CO [6]. In spite of its beneficial effects known complications include barotraumas and neurological associated oxygen toxicity which may manifest as generalized seizures [14]. Moreover we describe an unusual case of hyperkalemia which we speculate to have occurred as a result of the production of reactive oxygen species causing cellular damage.
Conflict of Interest

No conflict of interest was declared by the authors.

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